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CHANGING CONCEPTS OF THE ROLE OF TOBACCO IN THE MANAGEMENT OF DISEASE*

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FOR almost as long as people have been using tobacco, writers have been speculating on its role in the genesis of disease; and practising physicians, governed empirically by their clinical experience, or morally by their own feelings, or logically according to their premises, have been advising their patients to stop smoking, to cut down on their tobacco consumption, or even (in the old days when Tobacco was considered to have its therapeutic uses [see Silvette, Larson and Haag[205], 1958]) to indulge. Except for a few genuinely tobaccogenic diseases (of which the most unequivocal is tobacco allergy), the etiological role of tobacco is just as obscure today as, say, a century ago; but a survey of a considerable sample of the clinical tobacco literature of the past 50 years has revealed a significant change in opinion regarding

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the place of tobacco in the management of certain diseases. This latter is independent of speculation or opinion regarding tobacco etiology. Thus, there is no perceptible agreement among clinicians concerning the role of tobacco in the etiology of thromboangiitis obliterans, but there is virtual unanimity that tobacco exerts a harmful influence once the disease is established, and should therefore be strictly forbidden patients with this condition. In the present paper, we are concerned only with tobacco-use in the management of certain diseases; and we must emphasize, what is apparent from the preceding sentence, that, with the perhaps sole exceptions of tobacco allergies and nicotine hypersensitiveness, the observed influence of tobacco or tobacco-smoking on the course of any disease affords no valid information concerning its etiology.

One may venture to generalize that, where once smoking was categorically forbidden in many diseases, it is now permitted in moderation by competent authorities (Doran[50], 1948). However, this generalization is subject to so much qualification that it is quite useless, if not downright misleading; and, indeed, the whole point of this aspect of medical treatment is that it is not subject to generalization, and even this latter generalization must be qualified in the case of tobaccogenic disease. We had at first thought that a survey on historical principles might be revealing (as though the progress of therapeutics followed a uniquely straight and upward line, forsooth!); but on further examination, it appeared more advantageous to discuss in some detail the role of tobacco in the management of a few selected diseases: tobacco allergy, angina, and amblyopia; cardiovascular diseases; chronic bronchitis, pulmonary emphysema, and pulmonary tuberculosis; and peptic ulcer. These diseases have in addition the merit, or demerit, that they occupy a good deal of space in the tobacco literature, and so afford a sufficient body of opinion on which to comment.

TOBACCO ALLERGY. The existence of a true allergy to tobacco appears to be indisputable, but its prevalence is difficult to determine, perhaps because of its protean manifestations (Silvette, Larson and Haag[204], 1957). In deciding who is allergic to smoking, Rosen[179] (1954) considered as of paramount importance a "clinical entity" of signs and symptoms which cleared up completely on stopping smoking, and which could be reproduced by smoking. This observation sufficiently defines the place of tobacco-smoking in the management of tobacco allergy.

TOBACCO ANGINA. It is far from clear whether tobacco angina is a disease, or merely a set of signs and symptoms of nicotine hypersensitiveness or tobacco allergy; but, although resolution of this point would be of great theoretical and philosophical interest, it is fortunately not necessary here. In either case, the etiological relationship of tobacco is, by definition and diagnosis, evident; and no doubt the proper management of this condition entails abstinence from its precipitating cause (Scott[194a,b], 1952). Nevertheless, Hyman and Parsonnet[102] (1932) cautioned that instances will arise where the physician must not be too dogmatic in depriving a patient in whom tobacco may cause anginal pain of such comfort as he may secure from smoking. In certain cases, the soothing effect of a cigar, pipe, or cigarette now and then may be of decided benefit, while its complete withdrawal with subsequent discomfiture may be fruitful of such emotional stress that the latter may in itself harbor the necessary element leading to an anginal attack. These authors also stated that the great cardiologist,

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Sir James Mackenzie, had pointed out that a single cigar might do more good in certain cases than all of the drug sedatives at his command.

TOBACCO AMBLYOPIA. By definition of tobacco amblyopia, as Heaton, McCormick and Freeman[90] (1958) have pointed out, tobacco is a necessary factor; that is, it is not possible for non-users of tobacco to develop *tobacco* amblyopia. (The implications of this have been discussed in detail in an essay by the present authors entitled "Tobacco Amblyopia: The Evolution and Natural History of a 'Tobaccogenic' Disease [Silvette, Haag and Larson[206], 1960].) Insofar, then, as this disease is a true toxic amblyopia specifically due to tobacco, it would appear to follow that tobacco-use should be forbidden. Virtually all of the numerous writers on the subject have considered abstinence from tobacco to be either necessary or important or desirable (it would not be useful to categorize the writers themselves), in the successful treatment of tobacco amblyopia (see Silvette, Haag and Larson[206], 1960). Lautenbach[116] (1898) forbade smoking, but permitted chewing; Lillie[127] (1934) permitted the smoking of cigarettes. In many cases, abstinence from tobacco was supplemented by drugs or, more recently, by an enriched diet or vitamins. Regarding medication, Hambresin and Schepens[84] (1946) wrote: Everything helps, everything cures, from the moment the patient stops smoking. In some (rare) reported cases, however, tobacco amblyopia was not ameliorated by abstinence from smoking; in other cases, good recoveries were made in spite of continued smoking by the patients, especially following adequate vitamin therapy. Whether any of the reported cases were instances of pure and uncomplicated tobacco or nicotine amblyopia, it is, of course, impossible to say and hardly more possible to guess; and, by the same token, one cannot be sure whether the withholding of tobacco in these cases represents specific or non-specific treatment. So far as the well-being of the patient is concerned, however, the distinction is immaterial.

CARDIOVASCULAR DISEASE. Cardiovascular disease, like the other diseases to be hereinafter discussed, and unlike the tobaccogenic diseases just considered, is known to occur in non-smokers as well as in smokers; and so the question of tobacco etiology does not arise, even as a prerequisite to discussion of management of the patient (though writers without number and the power of logical thinking incessantly assert the contrary). In deference to the sincerity of their error, however, we shall state here that we propose to discuss the role of tobacco in the management of such diseases regardless of their etiology, whether really tobaccogenic or not.

With respect to established cardiovascular disease, published opinion has varied from the prohibition of smoking (Bornemann and Hochrein[31], 1955; among many others) to allowing smoking if the patient is not sensitive to nicotine (for example, Stroud[214], 1946), with all shades of opinion in between. Lee and Bryner[117] (1957), among others, considered that, although the patients should be advised to stop smoking, cessation may result in the substitution of obesity, chronic anxiety, or other equally stressful reactions, which the physician should try to prevent.

Writing of another category of patients, Brooks[34] (1915) thought it probably unwise to permit the use of tobacco in cardiovascular diseases when symptoms of cardiac embarrassment occurred. "In short," wrote Levy (in his discussion of Starr[212], 1951), "it is individual susceptibility rather than the

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presence of disease which determines the effects of smoking on the cardiovascular system." Regarding the argument that the euphoria engendered by smoking in the habitual smoker compensates for the milder cardiovascular changes caused by smoking, Ross and Ivy[181] (1946) have pointed out that this has not been applied, and cannot be applied with assurance to those persons who show marked cardiovascular reactions to smoking, that is, to hyperreactors. It is, perhaps, well to emphasize that hypersensitivity to nicotine, no less than tobacco allergy, is a sound contraindication to smoking in any disease.

HYPERTENSION. It has been said that tobacco is harmful in this condition (Lintz[129], 1933; Engel[58], 1935), and therefore some writers have stated that tobacco is contraindicated in hypertension (Gutierrez Muro[81], 1934; Hassencamp[89], 1939; Maris[136], 1944; Allanby[5], 1959). Owing to the great variation in the circulatory effects of smoking produced in different patients with arteriosclerosis and hypertension in their series, W. G. Thompson and Shelden[218] (1917) felt it desirable that each case be separately studied before giving rules for controlling the tobacco habit. All persons who have latent hypertension or hypertensive disease do not experience such increased vasospasm (increased blood pressure, constriction of retinal arterioles) following inhalation of tobacco-smoke, as Herrell and Cusick[93] (1938) found in hyperreactors; in fact, many of the former were found to have little or no change in blood pressure. These authors felt, however, that such patients have everything to gain, and nothing to lose, by discontinuing the use of tobacco.

Jones[106] (1941) pointed out that individual susceptibility to tobacco must be taken into account in considering the question whether hypertensives should smoke: what would cause serious trouble to some susceptible patients would not harm the majority. He recommended that excessive users of tobacco should completely abstain for a time, but then might resume smoking in moderation.

During a panel discussion on hypertension (J. Am. Geriat. Soc., 5, 471, 1957), de Takats advised against smoking in this condition in younger or middle-aged patients who still have flexible vessels which respond to nicotine stimulation; but if there is diffuse arteriosclerosis in a patient who has smoked all his life, and nicotine does not raise his blood pressure, the patient was not deprived of smoking. Edwards suggested that most patients with essential hypertension refrain from smoking for a month; if these patients feel better and their blood pressure is lower, they will usually not want to start again. Talso agreed with Edwards; however, if the patient returned at the end of a month without cigarettes, and was obviously nervous and tense, he considered it much better for such patients to smoke, since smoking relieved some tension. Shick was of the opinion that eliminating smoking has never hurt anyone with hypertension.

W. N. Thompson[219] (1940) suggested that tobacco be gradually deleted from the usage of hypertensive patients, presumably to mitigate the untoward effects of suddenly stopping the habit. However, when there is a considerable nervous element in the hypertension, smoking in moderation often has a soothing effect on the nervous system, and may be to some degree beneficial (Jones[106], 1941). In most cases of hypertension, smoking is contraindicated; in some cases, though, psychic tension may be relieved by limiting smoking (Med. Times, Manhasset, 76, 61, 1948). The question of

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the balance between what may be termed the pharmacological and the psychic effects of smoking in hypertension has been summed up by Moyer[156] (1957): Since smoking frequently aggravates hypertension, the habit should be dispensed with if it can be done without excessive psychic trauma; on the other hand, some patients have become so dependent on the smoking habit that the anxiety and psychic effect produced by discontinuing smoking would far outweigh the benefits to be derived therefrom. The decision is thus an individual problem to be made by physician and patient together. These same remarks have also been published by Moyer and Brent[157] (1959).

"HEART DISEASE." According to some writers, smoking must be forbidden in any type of heart disease (Brown[35], 1911; Green[79], 1950, illustrating the persistence of this belief), cigarette-smoking particularly (Green[79]). However, Brown would allow tobacco in certain very old confirmed tobacco-users who have developed a tolerance. Sir Thomas Lewis[124] (1937) thought it best that cardiac patients did not smoke at all, but if they will smoke, it must be done in strict moderation. This great authority pointed out that smoking is responsible, in a very large number of its habitués, for much strenuous coughing, and such coughs are most injurious to many "heart cases." In Levy's experience over a period of years, most patients with heart disease can smoke moderately without apparent harm (Levy *et al.*[123], 1947; Levy[121, 122], 1952, 1953). In view of the enjoyment afforded, and the feeling of satisfaction attained, it was Levy's (1952, 1953) opinion that the majority of patients with inactive forms of heart disease may therefore be permitted to smoke in moderation, while those sensitive to nicotine will fare better by abstinence.

"Patients with almost any form of heart disease are usually advised to abstain from the use of tobacco," wrote Neu[159] (1958), "although for a great many smoking provides pleasure and relaxation. There are certain situations, of course, in which tobacco should be interdicted, such as in congestive heart failure, acute myocardial infarction, and active rheumatic carditis."

CORONARY-ARTERY DISEASE. Although some writers have stated that tobacco should be strictly forbidden to all patients with coronary symptoms or disease (for example, Gaede[69], 1941; Olbert[163], 1955), the majority of observers have been less dogmatic. Some have felt that patients with disease of the coronary arteries should not, as a rule, smoke (Adlersberg[4], 1947; Study group on Smoking and Health[215], 1957), since "there are evidences that tobacco, and nicotine in particular, may have a harmful effect on the small percentage of persons who already have coronary-artery disease" (Hafkenschiel[82], 1957). Hilleboe[96] (1957) felt there was no evidence in the medical literature to support or deny the restriction against smoking for persons with coronary-artery disease; and Hunt[98] (1956) wrote that, in the present state of our knowledge, it is hard to believe that smoking in moderation always makes coronary disease worse.

In a report authorized for publication by the Council on Pharmacy and Chemistry of the American Medical Association, Ellis and Hancock[57] (1957) stated that the attitude of physicians in regard to restricting the use of tobacco or alcohol by patients with coronary-artery disease was frequently determined more by the moral or personal attitude of the doctor towards these substances than by any rational medical standards. There is no evidence, they noted that tobacco does the circulation any good, but some evidence suggests that it may produce mildly

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deleterious effects. Nevertheless, the physician should evaluate very carefully how important it is to restrict the happiness of a patient who is already limited in so many ways. Indeed, it is asking a lot of a man with coronary disease, who is so restricted, to give up the solace of tobacco (Wolferth[239], 1945). It seemed to this latter author that, in general, patients with coronary disease who stopped smoking do better than their less co-operative friends, but perhaps, he suggested, this is because they follow all their orders better.

Yater[246] (1955) thought that patients with known coronary-artery disease probably would do best not to smoke, unless they are made quite unhappy by the restriction. In the latter case, 10 to 15 cigarettes, or 2 or 3 pipes per day, should be the maximum; and heavy smoking, chain-smoking, and strong tobacco should be proscribed; nor should smoking be indulged in just before, during, or after effort (Lancet, 2, 861, 1955).

Conn and Kissane[42] (1958) stated that their patients with coronary disease are not told that they must stop smoking, except those who have peripheral arterial disease or who have angina associated with smoking. "The effects of nicotine on the arteries are explained to patients, and they decide for themselves whether to stop smoking or not. Many patients stop smoking with very little difficulty, but others have so many nervous symptoms when they are attempting to quit we feel they are harmed more by not smoking than by smoking."

CORONARY ARTERIOSCLEROSIS. Brooks[34] (1915) thought it highly probable that, when coronary sclerosis exists, the symptoms are accentuated by tobacco. Von Ahn[222] (1948) stated that everyday clinical experiences showed that many coronary sclerotic patients endure moderate tobacco-smoking without getting increased trouble; however, patients suffering from cardiosclerosis should be careful with tobacco, and not smoke at all if they get increased troubles from smoking. Ellis and Hancock[57] (1957) stated that it had not been proved that tobacco aggravates coronary atherosclerosis. Balotin[18] (1959) also declared that there is no proof that tobacco aggravates this condition; and he added, in words reminiscent of Wolferth[239] (above), that the physician should carefully evaluate the importance of restricting tobacco in a person who is already limited in so many ways.

ANGINA PECTORIS. After a review of the earlier literature, Mouriquand and Bouchut[155] (1912) concluded that, if tobacco can provoke crises of angina pectoris, these are ordinarily benign, and easily cured by the suppression of the toxic agent. Other authors have held that tobacco may induce paroxysms of angina pectoris (Blanc[25], 1925; Schlossmann[190], 1932; Lambert[114], 1935; Wilson and Johnston[235,236], 1939, 1941; Barnes[19], 1947; Pardee in Coffin *et al.*[41], 1956; among others); but it has also been said that precipitation of an attack by smoking is a rare occurrence (P. D. White[231], 1935; von Ahn[222,224], 1948, 1954; Levy[121], 1952), and then in patients with small coronary reserve (Graybiel, Starr and White[77], 1938) or in patients with coronary disease (von Ahn[224], 1954; Mendonca de Barros[147], 1956). Tests made by L. Wolff[240], (1937) indicated that tobacco did not, as a rule, alter the susceptibility to attacks of angina pectoris; and von Ahn[222] (1949), studying patients with cardiosclerosis and angina pectoris, pointed out how strikingly well these cardiac patients endured strong chain-smoking almost to the poisoning stage, in contradiction to the old idea that tobacco-smoking by angina pectoris patients would provoke or aggravate

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attacks. Kuo and Joyner[110] (1955) also noted that none of 11 patients with coronary-artery disease developed typical anginal attacks after test smoking. Von Ahn[222] (1948) concluded that moderate tobacco-smoking by angina pectoris patients who are not hypersensitive to tobacco cannot be considered an attack-provoking factor.

Since some patients with angina pectoris can smoke with impunity, while others have to give up smoking (Lambert[114], 1935), an unusual sensitivity to tobacco-smoke would appear to be involved in the case of those patients whose symptoms are precipitated or aggravated by smoking (Graybiel, Starr and White[77], 1938).

It has often been said that smoking may increase the severity or degree of pain in some patients with angina pectoris (Gallavardin[70], 1924; Halsey[83], 1933; Lambert[114], 1935; Brigden[33], 1958; among others). P. D. White[230] (1933), however, stated that most patients claimed smoking made little difference in influencing the frequency or severity of their angina pectoris.

It has been reported that the symptoms of true angina pectoris are not relieved by discontinuance of tobacco (Brooks[34], 1915; Smith[208], 1943). [Compare *tobacco* angina, above.] Other writers have reported that the frequency or severity of the attacks seems to be lessened if the use of tobacco is stopped (Halsey[83], 1933; Wolferth[239], 1945). Nevertheless, Wolferth himself noted that there are many heavy smokers with angina pectoris who are not improved to any preceptible extent by stopping the use of tobacco; and, according to Brigden[33] (1958), few patients find relief when the habit is dropped. Frequently, writers have referred to patients who have suffered angina pectoris only during periods of smoking, with cessation of attacks on omitting tobacco; but White and Sharber[232] (1934) pointed out that such cases have generally been few in number when specifically mentioned, and that no one physician had encountered many such cases. Rarely, in their own experience, did patients cease having angina attacks altogether when they stopped smoking.

A considerable number of writers have expressed the opinion that tobacco is contraindicated in angina pectoris (Potain[170], 1895; Rabinowitsch[175], 1922; Gallavardin[70], 1924; Romberg[178], 1925; Preston[174], 1929; P. D. White[230], 1933; Gutierrez Muro[81], 1934; Baltzan[14], 1939; Hassencamp[89], 1939; E. G. Scott[193], 1940; Clendening and Hashinger[39], 1941; Falk[62], 1947a, b; Barron[20], 1948; among others). Other writers have been less sweeping in their prohibition, allowing, if not sanctioning, the moderate use of tobacco (Sproull[211], 1935; Gilbert[73], 1936; among others). The prohibition of tobacco should rest on an individual basis (White and Sharber, cited by Master[137], 1935; Gilbert[73], 1936; von Ahn[222], 1948; among others). Dock[48] (1957) stated that 60% of patients with angina or healed myocardial infarcts show immediate evidence of impaired function following smoking, and are wise to abstain. A trial of abstinence is justified (R. B. Scott[194a,b], 1952). If a particular patient abstains from smoking for 3 months, and no relief occurs, he will have proved that tobacco is not injurious to him (Master[137], 1935); thereafter, the patient with will-power may smoke a few cigarettes a day (Mendonca de Barros[147], 1956).

R. L. Levy[120] wrote in 1934: "The attitude which the physician takes toward tobacco will vary. In general, patients with angina pain are better off without it. For many, it is a poison and its use results in increase in the frequency and severity of attacks.

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There are those, however, who derive real comfort and emotional stability from smoking. In them, curtailment is urged and the denicotinized cigars and cigarettes may be tried. The value of the de-nicotinizing process is open to question; but the patients themselves not infrequently think that they can smoke such brands with comfort whereas ordinary varieties are less well borne. Perhaps the reason for such experience lies in the fact that the de-nicotinized products, especially the cigars, contain milder tobaccos." Another authority, Master[138] (1956) wrote that 30 years ago, tobacco, even in small amounts, was taboo; today, tobacco is no longer banned in all cases; its proscription depends on its effects on the patient's well-being. There is little point in forbidding a tense patient to smoke a little, if that serves to relax him. Shepherd[196] (1947) considered it worse in most cases for patients with angina pectoris to worry about not being able to smoke, than it was for them to smoke.

It may be noted that the presence in a room filled with tobacco-smoke has been said to be usually as bad for the angina patient as smoking itself (Master[137], 1935).

According to Master[138] (1956), the view is no longer accepted that alcohol and tobacco in combination are good (alcohol dilating, tobacco constricting, the coronary vessels); it is now believed that a combination of alcohol and tobacco in less moderate doses actually aggravates the anginal syndrome.

Barnes[19] (1947) was cautious in concluding that he could advise patients with coronary disease (including angina) that smoking is not harmful to them. *Med. Times,* (76, 525, 1948) pointed out that there was at that time controversy over whether or not smoking should be forbidden in patients with angina pectoris. This controversy may be said to be still with us in the case of patients in the plural; but it would be scarcely correct to say that controversy exists in the case of the individual patient, in whom the clinical trial generally effects a resolution of the problem.

We may emphasize, with von Ahn[222] (1948), that angina pectoris is merely a clinical syndrome which tobacco is able to provoke or aggravate under certain circumstances, and that the effect of agitation and haste and heavy meals and late hours may add up to more than that of the tobacco. Von Ahn and Göhle[225] (1949) described a case of a 57-year-old male in whom attacks of angina pectoris were chiefly precipitated by tobacco-smoking and by meals; and electrocardiograms recorded during each of these attacks showed reversible changes characteristic of temporary insufficiency of the coronary arteries. Another case-report tells of a patient, a 39-year-old male, who had seizures of angina pectoris occurring spontaneously, as well as after exertion, emotion, exposure to cold air, eating, or smoking (Arai *et al.*[11], 1951). Mendonca de Barros[147] (1956) also noted that tobacco may release an anginal crisis, just as physical exertions and other factors can do; but the role of tobacco is much less important than that of the latter factors, since cases in which tobacco alone can precipitate an anginal crisis are rare, and, in the majority of cases, tobacco acts only in association with other factors.

ACUTE MYOCARDIAL INFARCTION. Wolferth[238] (1937) had no doubt that stopping tobacco-use lessened the incidence of pain in certain patients with coronary disease, but that, in a large number, no beneficial effect was observed. According to Stroud[214] (1946), eliminating tobacco will tend to lessen the frequency of attacks of substernal pain in healed coronary occlusion. Although

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Gertler and co-workers[71] (1951) found a greater percentage of smokers among patients with myocardial infarction than in a control group, and a significantly greater cigarette consumption in the former, they stated that the inference should not be drawn from these results that cessation of smoking would benefit or prevent coronary heart-disease.

Master[137] (1935) strongly believed that smoking, or even exposure in a room filled with tobacco-smoke, was harmful in coronary thrombosis. It has been stated that smoking 1 or 2 cigarettes could be an additional hazard in a patient with hypertension and myocardial infarction (J. Am. Med. Assn., 161, 301, 1956); and smoking was said to cause immediate evidence of impaired cardiac function in 60% of patients with angina or healed myocardial infarcts (Dock[48], 1957).

Smoking should be prohibited in the acute stages of cardiac infarction (Burch[37], 1949; Levy *et al.*[123], 1947; Levy[121,122], 1952, 1953; Pollack[168], 1956); and some authorities have stated that smoking is probably best avoided thereafter, especially in the nicotine-susceptible individual (Pratt[172], 1951; Dock[48], 1957). Pratt (1951), indeed, considered that all smoking should be permanently eliminated in the management of the post-thrombotic syndrome; even one cigarette may produce spasm, and the possibility was said to exist that nicotine may initiate thrombitis without spasm.

In a panel discussion on the clinical management of the acute stage of myocardial infarction moderated by I. S. Wright[245] (1957), Sprague, Ernstene, and Friedberg all stated that they required their patients to stop smoking; Stamler also was convinced that, with few exceptions, the sound procedure was to do likewise. Wright himself remarked: "We do not take pleasures away from patients for the fun of it. We only take away something that a man cherishes as much as his cigarettes, if we are convinced it is for his own good and may influence the outcome of a serious condition."

In an article provocatively titled "Danger! Doctors at Work," Forbes[65] (1958) wrote: "An example of [the physician's] over-care is the patient with a mild coronary thrombosis who is confined to bed for three months and afterwards forbidden to smoke, drink, or engage in his favorite recreations—all quite unnecessarily." This attitude characterizes the rather recently developed point of view that a dogmatic prohibition of smoking may not be in the best interest of all patients. "Since tobacco is known to cause arterial vasoconstriction and since, occasionally, electrocardiographic changes of coronary insufficiency can be seen to occur in sensitive persons after smoking, the use of tobacco would seem contraindicated after coronary thrombosis" (J. Am. Med. Assn., 167, 2159, 1958). "From a more practical standpoint, however, in the management of the entire patient it is at least equally important that he be emotionally rehabilitated, with the realization that his life can again be productive after a heart attack. If such rehabilitation requires that he be permitted to smoke a few cigars daily, it is probably more important that this be done than that the possible deleterious physiological effects be permitted to weigh too heavily. In general, moderation and not total abstinence seems the most practical answer. Most persons who have had a complete functional recovery after coronary thrombosis may be permitted a drink of liquor and a few cigars daily if that was their life-long habit previously." Also concerning smoking after a coronary thrombosis, Hunt[98] (1956) wrote:

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"If smoking causes severe coughing, especially at night, that is a good reason for stopping it altogether. But to stop smoking is sometimes a mixed blessing, quite apart from the mental irritability it often causes: when some patients give it up their appetites improve, they take larger meals, and eat sweets instead, and the increase in weight soon does more harm than smoking did before. To prevent further infarcts, it seems clear that it is better to keep patients underweight rather than overweight. In the present state of our knowledge, it is hard to believe that smoking in moderation always makes coronary disease worse."

PERIPHERAL VASCULAR DISEASE. The authorities are virtually in agreement that tobacco is harmful in individuals with peripheral vascular disease, and should therefore be prohibited (I. S. Wright[242,243], 1933, 1940; Gutierrez Muro[81], 1934; Lampson[115], 1935; Ochsner and DeBakey[160], 1939; Chapman[38], 1941; Tecoz[216], 1941; Herbig[92], 1942; Evans and Stewart[61], 1943; Pratt[171], 1946; Hendrick and Aycock[91], 1947; Lilly[128], 1949; DeCamp[45], 1949; Levy[121,122], 1952, 1953; R. B. Scott[194a,b], 1952; G. N. Pratt[173], 1957; among others). Lilly (1949) refused to treat a patient with peripheral vascular disease who could not discontinue smoking, a policy also followed by DeCamp (1949). Chapman (1941) stated that use of tobacco must be discontinued entirely if satisfactory improvement is to be expected in the treatment of occlusive peripheral vascular disease; Hendrick and Aycock (1947) stated that patients with such disease should not smoke if they expect to keep their limbs.

When tobacco hypersensitivity or idiosyncrasy is demonstrated either clinically or by positive skin reactions or by both, exclusion of tobacco should be one of the prerequisites in treatment of patients with peripheral vascular disorders (M. A. Green[78], 1942). Whether tobacco be a specific allergic excitant or a potent nonspecific irritant, it should be avoided by all persons who suffer from vascular disease (Maurer and Spain[140], 1954).

Although, in susceptible individuals, tobacco should be absolutely interdicted, on the other hand, Boyer[32] (1942) thought it possibly unwise to attempt abrupt modification of deeply ingrained habits, especially in the elderly. It has been remarked that tobacco is safer after the age of 60 years (Tuohy[220], 1943).

Denicotinized cigarettes of any type should also be avoided in the presence of peripheral disease, since these, as well as regular cigarettes, cause decreases in peripheral blood flow (Evans and Stewart[61], 1943).

INTERMITTENT CLAUDICATION. According to Schlesinger[187,188,189], (1911, 1912, 1921), patients with intermittent claudication are absolutely intolerant to nicotine, and even moderate smoking will revive symptoms which have disappeared. Years later, DiCio and Guerra[47] (1940) also noted that many patients who no longer suffered attacks of intermittent claudication had renewed crises if they resumed the use of tobacco. Complete suppression of the use of tobacco sufficed to produce recovery in a large number of the latters' patients whose symptoms did not exceed 2 years' duration. According to Lian, Puech and Viau[125] (1927), however, suppression of tobacco consumption did not prevent the disease from continuing to manifest itself.

John[105] (1913) and Hassencamp[89] (1939) both stated that smoking should be forbidden in intermittent claudication. A recent assessment of the position has been made by Wessler[228] (1956), who noted that the

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continued use of tobacco by patients with this disorder is still strongly interdicted by many authorities. The fact that tobacco-smoking increases the tone of peripheral vessels is adequate justification for urging all patients with acute arterial insufficiency to stop smoking, at least until the imbalance between obstruction and collateral flow has been improved. However, for the large group of ambulatory patients with intermittent claudication from atherosclerosis, the need for such restriction is not so clear. There is no evidence that tobacco, any more than mildly cold weather, adversely affects the course of the disease. Some investigators have been unable to demonstrate any deleterious effects of tobacco on intermittent claudication due to atherosclerosis. Others have permitted patients to smoke a few cigarettes daily without apparent harm. For many of these individuals, tobacco-smoking is a well-trenched habit, and abstinence may be neither necessary from the organic point of view nor desirable for emotional reasons. Wessler no longer insisted that patients with stable intermittent claudication secondary to atherosclerosis discontinue tobacco as part of the therapy of their peripheral arterial insufficiency.

THROMBOANGIITIS OBLITERANS. With a unanimity rare, if not unique, among writers on tobacco, virtually all observers and commentators are agreed that smoking is harmful in thromboangiitis obliterans (TAO), and that all patients with this disease should abstain from tobacco (Silbert[198,203], 1926, 1946; Allen and Brown[7], 1928; Miller and Kaufman[150], 1928; Barker[16,17,18], 1931, 1933, 1934; Samuels[184,185,186], 1931, 1934, 1936; Maddock and Coller[133], 1933; Lampson[115], 1935; Horton[97], 1938; McKittrick[145], 1939; Wright[241], 1940; Clendening and Hashinger[19], 1941; Theis and Freeland[217], 1942; Hildenbrand[95], 1945; Lowenstein[130], 1945; Allen, Barker and Hines[8], 1946; Goodman, Messinger and White[75], 1946; Freeman[67], 1947; Shepherd[190], 1947; Bloom[26], 1948; DeTakats[46], 1948; Lynn and Burt[131], 1949; Manoil[134], 1949; Mead[146], 1949; Rosenauer[180], 1950; Wessler[228], 1956; Matousek[139], 1957; among others). The abstinence must be complete and permanent. Many patients continue to smoke in spite of repeated warnings that it will result in the loss of a leg; in part, this is due to unwillingness to believe that they have a special susceptibility to tobacco which most people do not share, and in part to the attitude of physicians who disparage the advice of experts in this field, and who suggest that it will be sufficient to reduce the amount of smoking (Silbert[203], 1946). Some writers have gone so far as to state that, if the patient does not cooperate in stopping smoking, the physician should give up the case (Mead[146], 1949; Matousek[139], 1957).

That abstinence from tobacco is beneficial, and should form the primary basis for all treatment in TAO, wrote Gifford and Hines[72] (1951), is not hypothetical, but is a well-established fact. Silbert[198,199,200,201,202,203] (1926, 1927, 1930, 1935, 1938, 1946) has repeatedly emphasized that cessation of smoking is the most important therapeutic measure, and that the disease seldom progresses as long as the patient refrains from smoking. McGovern[143] (1947) has also stated that patients with TAO who stop smoking can be assured a remission in their disease, which can and does last for years with no need for any form of therapy other than abstinence from nicotine. Case-histories illustrating such cures have been reported (for example, by Gifford and Hines[72], 1951). Though treatment of TAO is said to be useless without complete abstinence

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from smoking (Samuels[186], 1936; Hildenbrand[95], 1945; among others), discontinuance of the use of tobacco without other measures was insufficient to bring about lasting improvement in symptoms (Bernheim and London[24], 1937). Marchak[135] (1933) has stated that he had never ameliorated a case of TAO in which the patient continued to smoke during treatment, but had never dared counsel a patient to cease smoking as a sole form of treatment; hence, he could not say whether or not absence of tobacco intoxication by itself would be sufficient to check the disease. Saland[183] (1938) pointed out that one must be careful in evaluating the tobacco-smoke factor in TAO; for, while improvement has been reported following cessation of smoking, one must remember that other methods of treatment had been instituted at the same time.

The proscription applies to tobacco in any form (Lowenstein[130], 1945; Goodman, Messinger and White[75], 1946), and to denicotinized cigarettes (Mead[146], 1949; Wright[244], 1954) and filtered cigarettes (Wright[244], 1954) as well. It would seem, however, that if the cause of the exacerbation of the disease is vasoconstriction resulting from nicotine, as many have postulated (Maddock and Coller[133], 1933; Lampson[115], 1935; Ross and Ivy[181], 1946; Ejrup[56], 1948; among others), then prohibition of de-nicotinized or filtered cigarettes, or both, must be made on the basis that neither the denicotinizing process used nor the filter employed removes sufficient of the nicotine in the smoke. If, on the other hand, the mechanism by which the symptoms are produced or aggravated is an allergic one, as other writers suspect (for example, Harkavy[85], 1957), then it follows that even complete removal or filtration of non-antigenic nicotine would be useless. This raises the important practical point that, unless one knows which of the many ingredients in the chemical complex known as tobacco-smoke is the etiologic, or aggravating, agent, any attempt to remove one of the ingredients from smoke without reference to the others must rest upon an unsound scientific foundation. However, even in the absence of such exact knowledge, the unanimous condemnation of smoking in cases of TAO (J. Am. Med. Assn., 119, 534, 1942) appears to be thoroughly justified.

Any form and stage of TAO is exacerbated by the use of tobacco (Rosenauer[180], 1950), although there appear to be numerous exceptions even to this rule (Horton[97], 1938). In many cases, smoking may not only augment symptoms, but perhaps also hasten pathologic changes (Barker[17], 1933; Brit. Med. J., 2, 1149, 1936; Abramson[3], 1944), and eventually lead to gangrene (Abramson, 1944). Barker[16] (1931) has published data indicating that the disease is likely to run a considerably more malignant course if patients have used tobacco excessively; and nearly half of Silbert's[202] (1938) patients who required amputation had persisted in smoking in spite of many warnings to stop (see also Silbert[203], 1946).

The course of TAO may to some extent be arrested by the avoidance of smoking (Brit. Med. J., 2, 1149, 1936); and it has been said that complete abstinence from cigarettes can arrest TAO before amputation becomes imminent (Eastcott[52], 1959). The disease is often checked on stopping smoking, and recurs on resuming the habit (Sigler[197], 1925; Silbert[199,200], 1927, 1930; Hildenbrand[95], 1945; Rosenauer[180], 1950). The disease seldom progresses as long as the patient refrains from smoking (Silbert[198,199,200,201,203], 1926, 1927, 1930, 1935, 1946), and there is no recurrence without resuming smoking

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(Silbert[200,203], 1930, 1946; Clendening and Hashinger[39], 1941). There is no lasting improvement without cessation of smoking (Bernheim and London[24], 1937; LeFevre[119], 1942; Abramson[3], 1944); or at least, patients who continued to smoke do not ordinarily improve as much as do patients who cease smoking (Allen[6], 1939).

Summing up his experience with 1,200 patients with TAO, Silbert[202] (1938) stated that individuals who continued to smoke were difficult to improve, and frequently grew worse in spite of treatment; while cessation of smoking alone was often sufficient to cause relief of symptoms and improvement in circulation in early cases. Patients who had been restored to good condition remained in excellent health without recurrence of symptoms, so long as they refrained from further use of tobacco. When non-smoking patients who had been in excellent condition for many years resumed the use of tobacco, recurrence of symptoms was the rule. No patient restored to good condition had ever had recurrence of trouble or required amputation, unless he had resumed smoking (Silbert[203], 1946).

Sirota[207] (1943) reported successfully treating cases of TAO and arteriosclerosis obliterans with deoxycorticosterone acetate, the patient meanwhile being allowed to continue smoking. Fisher[64] (1949) treated 12 TAO patients with tetraethylammonium chloride to the exclusion of all other therapy; those who smoked previously continued to smoke while under observation; and after 6 months, 8 patients were said to be symptom-free, except for a mild amount of intermittent claudication. In a preliminary communication, Eisen[55] (1958) reported effects of a new potent combination of vitamin A, niacin, and riboflavin (called ANR formula) applied for 1 to 10 years in a series of 17 cases of peripheral-artery disease, including 2 cases of TAO; 16 were heavy cigarette-smokers and one a non-smoker. In 14 of the patients, there was an improvement in 2 to 4 weeks, and improvement was marked in 3 to 6 months, whether the patient smoked or not. Withdrawal of ANR combination brought about prompt return of the initial syndrome, even when the patients were not smoking. The above observations suggest at least the possibility that the precipitating or aggravating effect of smoking on patients with TAO may be exerted secondarily to some more fundamental mechanism. Practical clinical management, however, can rarely wait upon the full elucidation of mechanism of action, and it is difficult to perceive a common fundamental mechanism in the respective therapeutic experiments of Sirota, Fisher, and Eisen.

CHRONIC BRONCHITIS. It is generally considered that the proper treatment of chronic bronchitis involves cessation of smoking (Greene and Berkowitz[80], 1954; Clough[40], 1956; Anderson[9], 1957; Menefee[148], 1957; among others). The prohibition of smoking in this condition ranges from Palmer's[164] (1954) suggestion that the patients should be encouraged to give up smoking, to Matousek's[139] (1957) statement that, if the patient with intractable chronic bronchitis will not stop smoking, the physician should divorce himself from the case.

The aggravation of chronic bronchitis by heavy smoking appears to result from (nonspecific) irritation from tobacco-smoke (R. B. Scott[194a,b], 1952; Menefee[148], 1957), and, not surprisingly, is correlated with inhalation of the irritant (Greene and Berkowitz[80], 1954; Clough[40], 1956). Thus, "smoker's bronchitis" was found to be rare in cigar- and pipe-smokers, who do not inhale, while the incidence-rate

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rose progressively with the quantity and duration of cigarette-smoking (Greene and Berkowitz). The smoker who does not inhale, or who smokes moderately, seems to suffer relatively little; but, Clough added, few maintain their smoking at such a level indefinitely, and very few can abstain from inhaling, unconsciously if not deliberately.

The relative importance of smoking as an irritant of the respiratory mucous membranes can be determined precisely only after further observations (Clough[40], 1956). However Menefee[148], (1957) held that tobacco-smoke was perhaps the greatest and most frequent source of irritation; and Greene and Berkowitz[80], (1954) estimated that smoking is 4 to 7 times more common as a cause of bronchitis than all other causes combined.

It may be noted that 21 of the patients with chronic bronchitis studied by Leese[118] (1956) claimed that the early-morning cigarette was a useful expectorant. Barach and his colleagues[15] (1952) had previously found, in certain patients with bronchial asthma and pulmonary emphysema, that coughing due to inhaling cigarette-smoke resulted in expectoration of considerable amounts of bronchial secretions and in a marked relief of dyspnea.

PULMONARY EMPHYSEMA. Franklin and co-workers[66] (1958) stated that evidence has been presented in the literature that heavy smoking, especially cigarette-smoking, is a common and perhaps a specific cause of obstructive pulmonary emphysema. Actually, such evidence is relatively scanty and unconvincing, although opinion to this effect is plentiful; but, whatever the etiological relationship between smoking and emphysema, smoking is generally held to be harmful to patients with this disease. Most writers are agreed that, in the treatment of pulmonary emphysema, cessation of smoking is very important (Gutierrez Muro[81], 1934; L. Hyde and B. Hyde[101], 1956; Matousek[139], 1957; Peters and Prickman[165], 1957; Mayock[141], 1957; E. A. Brown[36], 1958; Motley and Kuzman[154], 1958; among others). Persons with bronchitis who develop the earliest signs or evidence of emphysema should stop smoking in any or all forms; and Menefee[148] (1957) bluntly told his own patients: "Make up your mind whether you would rather smoke or breathe." If, however, after 30 days of no smoking, the emphysematous patient feels no better; Brown[36] (1958) allowed him to smoke in moderation. "In our patients with emphysema," wrote Bedell and Seebohm[21] (1959), "we recommend that they stop smoking if possible. We do not demand that they do so. Some patients are happier if they can still smoke a few cigarettes a day, and we have been willing to make this compromise."

In a large number of instances in a series of 250 cases, patients with pulmonary emphysema who succeeded in stopping smoking obtained complete subjective relief from cough and wheeze, and frequently wheezing was decreased or cured (Abbott *et al.*[2], 1953). Conversely, smoking tends to aggravate the increase in air-way resistance so characteristic of chronic pulmonary emphysema (Eich, Gilbert and Auchincloss[54a,b], 1956; Attinger, Goldstein and Segal[12], 1958).

PULMONARY TUBERCULOSIS. A number of writers have expressed the opinion that smoking is harmful in active tuberculosis, and should be forbidden (Jankau[104], 1894; Herrick[94], 1931; Lickint[126], 1935; Kurova and Nezlin[112], 1936; among others). Although they stated that no dependable data are at hand regarding the role of smoking in tuberculosis, Kupka and Breslow[111] (1957) suggested an explanation of

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why it might be harmful; respiratory trauma, which is inevitable in a coughing, compulsive smoker, probably decreases the ability of pulmonary tissue to defend itself against the tuberculosis bacillus. Other authorities have considered that, at best, smoking can do no good, and, at worst, may unfavorably influence the development of the disease (Bogen[27], 1937; J. Am. Med. Assn., 128, 1132, 1945; *ibid.* 144, 437, 1950; Rickmann[177], 1953; Shah *et al.*[195], 1959); while still others (J. A. Miller[149], 1934; Ebers[53], 1948; Wiese[233], 1954; Winkelmann[237], 1957) have felt that, within limits of moderation, the lesions of pulmonary tuberculosis do not appear to be worsened by the use of tobacco.

Pointing out that there is no special clinical picture of "smoker's tuberculosis," as there is of certain other types, Ebers[53], (1948) noted that the problem of whether smoking injures those with pulmonary tuberculosis has by no means been decided. He himself recommended individualized prohibition of smoking in certain cases, but considered that a general rule forbidding tuberculous patients to smoke was psychologically unwise. A similar point of view was expressed by Winkelmann[237] (1957), who compared results of the cure of 1,000 male smoking and nonsmoking patients at the Jerichow Tuberculosis Sanitorium, but was unable to establish an untoward influence of tobacco-smoking in tuberculosis. Accordingly, he suggested the discontinuance of a blanket smoking prohibition, as practised in various sanitoria during cures.

Schwartz[192] (1945) obtained the opinions concerning smoking in tuberculosis from about 50 directors of sanitoria: 13 thought smoking definitely harmful to tuberculous patients; 36, harmful in excess; 1, not harmful. Smoking was completely forbidden by 8; 16 permitted smoking on the recommendation of a physician; 20 permitted smoking, but without approval; 6 did not forbid smoking. Nine attempted to control smoking by advice; 20, by specifying certain places at certain hours for smoking; 7 used threat of discharge; 11 did not enforce their rules; and 3 had no rules. Although more than 95% of the directors felt that smoking was at least injurious, less than 20% felt it should be completely forbidden, this latter attitude perhaps engendered by the fact that it is quite difficult to discourage patients from a habit of long standing in return for a benefit of questionable value. Wiese[233] (1954) circulated a questionnaire in regard to tobacco-smoking for tuberculosis patients, and received replies from 19 chief sanitorium physicians: 7 were outspoken for generally prohibiting smoking; 9 were in favor of prohibiting smoking in the sick-rooms and rest-corridors; one was against smoking in the acute process; one judged according to the particular condition; one prohibited smoking for bed-ridden patients. Wiese himself thought that one cigarette or cigar would not "overthrow" the tuberculosis patient, but that it was better during therapy to discontinue smoking. Strict prohibition was not completely successful; physicians and personnel should set the example, and not smoke.

PEPTIC ULCER. When McHardy and his colleagues[144], (1959) recently stated that smoking was a controversial issue in the management of acute peptic ulcer, we may understand them to mean simply that a great number of writers have expressed a wide variety of opinions on the subject. For controversy ["*Controversy* is worse than an unprofitable task; instead of eliciting truth, it does but expose the failings of the parties engaged."—

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Crabb's *English Synonymes; "Controversial . . .* suggests provocativeness, violent differences of opinion, and sometimes the activities of propagandists."—Webster's *Dictionary of Synonyms*]—for "controversy" is happily rare among those interested in tobacco (the cigarette-smoking-lung-cancer *controversy* is an exception which only too sadly justifies the definitions of "Crabb" and "Webster"); but differences of opinion in this field are so common that only *unanimity* of opinion (as in the case of thromboangiitis obliterans) can astonish us. This digression into definition is not without purpose or importance, for a controversy may be resolved by the efforts of knowledgeable and objective judges, whereas differences of opinion among a swarm of writers can only operate as a springboard for further opinion, not as a basis for rational judgment.

With respect to the role of tobacco in the management of peptic ulcer, the clinical opinions expressed may be (more or less arbitrarily) separated into three main categories; by writers in the first category, any tobacco-use is more or less flatly forbidden; by writers in the second group, excessive use is discouraged rather than forbidden, and moderate use of tobacco permitted, either openly or tacitly; and by the third group, tobacco-use is regulated on an individual basis.

Tobacco was said to be contraindicated or prohibited in peptic ulcer (during treatment or forevermore) by the following writers, among others: Rehfuss[176] (1927); Bennett[22] (1928); MacLean, Jones and Fildes[132] (1928); Finkelstein[63] (1930); Hartman[88] (1930); Jordan[107] (1930); S. Harris[86] (1930-31); Ogilvie[162] (1935); Ochsner, Gage and Hosoi[161] (1936); Davis[44] (1939); Spriggs[210] (1940); L. A. Smith and Rivers[209] (1943); Morlock[152] (1944); Portis[169] (1944); Twiss and Parsounet[221] (1945); Wilkinson[234] (1948); Weinstein[226] (1951); Olbert[163] (1955); Harrison[87] (1959); and G. McHardy, R. J. McHardy and Browne[144] (1959). This prohibition extended to denicotinized as well as to regular tobaccos (Bernay and Faure[23], 1937; Couturat[43], 1937).

Restricted use of tobacco was permitted, or at least not uniformly forbidden, by others (F. W. White[229], 1919; Hurst[100], 1923; Eusterman[59], 1925, 1926; Rowlette[182], 1928; I. Gray[76], 1929-30; T. Hunt[98], 1937; Aaron[1], 1945; Planck[167], 1946; Glatzel[74], 1947; among others).

Although he warned in all cases against immoderate smoking, Rowlette[182] (1928) recommended studying the reaction of the ulcer patient to tobacco; and Kirsner[108] (1958) agreed that the use of tobacco in such patients is dealt with most practicably on an individual basis. To the latter writer, moderate smoking seemed without harm in many instances; excessive smoking, on the other hand, is undesirable. The recommendation of complete abstinence is preferable to an ineffectual suggestion of "decreasing" the quantity of tobacco. Excessive smoking ordinarily reflects increased nervous tension; the important problem, therefore, is relief of the emotional stress. Ordinarily, wrote Boles and Dunbar[30] (1946), tobacco is to be avoided by the ulcer patient of younger years; however, harm from its temperate use by a person past 60 is hard to imagine, considering the lowered acidity of his stomach, and the peace of mind its use affords after years of temperate indulgence. Morlock[153] (1958), noting that for the most part ulcer patients are unwilling to discontinue their use of tobacco, thought some compromise ordinarily must be made in regard to this factor.

Even moderate amounts of tobacco

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may be harmful in susceptible individuals (Eusterman[59], 1925, 1926). Patients with peptic ulcer who are hypersensitive to tobacco will experience aggravation of their symptoms by the use or overuse of tobacco, and such persons should be enjoined to give up smoking (Morlock[153], 1958). Whether such "susceptibility" refers to nicotine hypersensitivity or to tobacco idiosyncrasy on the part of the patient is not always clear. Andresen[10] (discussing Roth, Ivy and Atkinson, 1944) stated that he rarely found smoking harmful in ulcer patients, and that these cases were probably explicable on an allergic basis. However, most writers have apparently taken it for granted that nicotine is the active and implicated ingredient of tobacco-smoke.

Regarding the time as well as the degree of tobacco limitation in patients with peptic ulcer, S. Harris[86] (1930-31) felt it wise to instruct ulcer patients to discontinue the use of tobacco for the rest of their lives. After recovery from peptic ulcer, Spriggs[210] (1940) would allow one cigarette after a meal; Douthwaite[51] (1947) would limit smoking to a pipe after breakfast and another after dinner throughout the life of a chronic ulcer patient.

Such writers who have commented on this point appear to be agreed that moderate smoking after meals is less harmful to the ulcer patient than is smoking when the stomach is empty (Rowlette[182], 1928; T. Hunt[99], 1937; Brit. Med. J. 2, 1098, 1939; Morlock[153], 1958).

Although there is a considerable volume of (very conflicting and confusing) evidence concerning the effect or non-effect of smoking on gastric acidity and secretion, the great majority of medical writers has used clinical criteria in assessing the influence of tobacco on the course of the patient's disease. Thus, I. Gray[76] (1929-30), observing that clinical improvement in some patients with ulcer occurs only after cessation of smoking, remarked that the therapeutic test, and not the chemical and roentgenographic findings, is the criterion as to whether or not the individual should smoke. Schnedorf and Ivy[191] (1939) stated, with respect to the routine prohibition of the use of tobacco by ulcer patients, that one apparently cannot rely on clinical impressions, because the views expressed vary so widely. It is, however, difficult to see just what the physician can substitute for clinical impressions in these cases, and support for this view is afforded by Douthwaite's[51] (1947) sound comment: "Experimental evidence favours harmlessness of smoking to patients with chronic ulcer. Neither rate of emptying of the stomach nor acidity can be shown to be appreciably affected. As against this, it is a spontaneous observation of countless sufferers from duodenal ulcer that the pain, in the active phase of the disease, will always be produced or, if present, aggravated by the smoking of a single cigarette. Again, on inquiring into the change of habits preceding a relapse, how often does one find that the only prophylactic measure which has lapsed has been abstinence from tobacco." Indeed, Ivy[103] (1944) expressed himself as uncertain why so many ulcer patients improved after giving up smoking, which is at least a tacit recognition of the reliability of "clinical impression".

It has been said by some writers (for example, Boles[28,29] 1943, 1948) that tobacco is a major influence in the prevention of healing and the recurrence of peptic ulcer, and by others (for example, R. B. Scott[194a,b], 1952) that smoking apparently does not affect healing of the lesions. Outside the realm of opinion, Doll, Jones and

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Pygott[19] (1958) tested the effect of smoking on ulcer healing (reduction in size of the ulcer) in patients with gastric ulcers who were regular smokers, and, from their results, concluded that, in some patients, smoking interferes with the healing of a peptic ulcer, and helps to maintain its chronicity. It may also be mentioned that Piper and Raine[166] (1959), after demonstrating that gastric-juice volume, total and free acid, and chloride were significantly increased by smoking, concluded that such a degree of gastric stimulation would be unlikely to fail to influence peptic ulceration, and that their investigation supported the clinical observation of Doll and his colleagues that smoking delays the healing of peptic ulcers.

Friedrich[68] (1934) stated that smoking is contraindicated in patients who have been operated on for gastric ulcer; Moynihan[158] (1923) advised patients not to smoke for 3 months after operation, and then always in strict moderation; Lahey[113] (1945) warned that any patient who has had either radical or conservative surgery for peptic ulcer should adjust his eating, drinking, and smoking habits for the lowering of gastric acidity in order to lessen his chance of having another ulcer develop. In a recent study of subtotal gastrectomy in 115 smokers and 56 non-smokers, Mitty, Rousselot and Delany[151] (1959) found that patients who smoked postoperatively had no more significant digestive complaints than the non-smokers; however, more major late complications occurred in the smokers.

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So far, for the most part, we have discussed, or, rather, canvassed opinion concerning, the role of tobacco in the management of disease; and we have now to turn our primary emphasis to the influence of tobacco-use on the patient himself. This latter consideration falls into two categories: the effect of smoking, and the effect of ceasing to smoke.

There has long existed the realization that, so far as the smoking patient or person is concerned, one must allow for a balance between the "psychic" benefit derived from smoking, and its "physical" harm (Boston Med. & Surg. J., 161, 231, 1909); also, between the individual pleasure derived from smoking, and the "increased mortality" of smokers often assumed or suggested by statistics (Wells[227], 1947); between the "tranquilizing" effect of smoking (Kitchen[109], 1916; Lancet, 1, 339, 1947; J. Am. Med. Assn., 138, 652, 1948; Levy[121], 1952; Lee and Bryner[117], 1957; Lancet, 2, 250, 1958; McArthur *et al.*[142], 1958), and the nervous stress which often accompanies giving up smoking for even the most compelling of reasons. In many individual patients, or classes of patients, as some of our authorities have pointed out, the beneficial effects of continuing to smoke may outweigh the manifest advantages of stopping smoking; and the decision is to be made, not only on the basis of the patient's disease, but with due consideration of the patient-as-a-whole.

From still another point of view, it may be said that smoking may prevent certain permanent undesirable effects of stopping smoking, for example, the danger of neurosis developing in individuals who find themselves unable to give up their habit (Stoll[213], 1955), or the danger that the increase in eating and body weight which often occurs following giving up smoking may place more "strain" on the heart than the smoking itself (*Med. Proc.*, Johannesburg, 3, 469, 1957). Some psychogenic smokers, upon giving up smoking, become psychogenic eaters instead (Lancet, 2, 282, 1957); the menace of obesity

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in ex-smokers appears to be real and to be causing some recent medical concern; and it is a so far unresolved question whether more persons are engaged in digging their graves with their teeth or with tobacco.

As for the purely temporary discomfort of "withdrawal symptoms" on stopping smoking, this should not be made the basis of any joint physician-patient decision whether the patient shall stop or continue his smoking. The physician's part in this decision has been set forth by Clough[40] (1956): It is our duty as physicians to inform our patients as fully and clearly as possible as to the facts as they are now known, and of the potential risks of smoking, and to let the patients make their own decision, as most of them will do anyway. Positive advice would depend upon the individual's physical condition, his smoking habits, and his reaction to smoking. Clough's advice presupposes that the physican shall keep himself fully and clearly informed as to the facts of the matter, which the latter cannot always find time or medical journals to do.

The main conclusions to be drawn from this survey on the role of tobacco-use in the management of disease are clear enough. In the case of specific tobaccogenic diseases properly diagnosed, the *cause* of the disease, by definition, tobacco, may be forbidden without doubts and should be prohibited without equivocation. If we define (as it appears we must) a non-tobaccogenic disease as one which occurs indistinguishably in both tobacco-users and non-users, regardless of its respective incidence-rates, then we may conclude, with respect to tobacco-use in the management of non-tobaccogenic but tobacco-aggravated diseases, that there *is* nothing better than abstention from tobacco, though sometimes there *may be* something worse: emotional disturbances of one sort or another, or the substitution of an even less desirable habit (for example, overeating) for that of moderate tobacco-smoking. But, as we have seen, these latter conclusions are often swept aside by a realization that the "psychic" good his habit of tobacco-smoking does a particular patient may be greater than its "somatic" harm. This (on the whole) fairly recent realization on the part of the physician represents the "changing concept" of our title: with tobacco-smoking, as with much else in the practice of medicine, the categorical imperative must sometimes yield to individual considerations.

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RADIOLOGY

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ROENTGENOGRAPHIC MANIFESTATIONS OF THE CARTILAGINOUS DYSPLASIAS

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A GROUP of similar skeletal diseases manifested primarily as a severe growth disturbance of enchondral bone formation has been described, particularly in the radiological literature. These cartilaginous dysplasias are often difficult to evaluate and classify, and, in the minds of many, considerable confusion exists in the differential diagnoses. In the majority of cases a careful personal and family history and physical examination, as well as laboratory and Roentgen findings, are necessary to arrive at a plausible answer and even then a definitive categorization of the skeletal dysplasia cannot always be made.

In this review of the literature, I shall present the classical findings of the more important of these diseases as observed on the roentgenogram. When certain criteria are applied, they will, in most cases, permit one to arrive at the most likely diagnosis.

The conditions described in this paper are listed in categories and will be discussed separately along with pertinent roentgenographic examples. Other skeletal diseases should perhaps be included, however, the study will be confined to only those which are unequivocally cartilaginous dysplasias and which often present similar and overlapping Roentgen aspects. All of these diseases are generalized in the sense that they affect the cartilaginous precursors of bone in a widespread manner.

The conditions covered are: 1) achondroplasia; 2) osteochondrodystrophy a) Morquio type, b) Hurler type, c) Leri type; 3) dysplasia epiphysalis multiplex; 4) dysplasia epiphysalis punctata; 5) dyschondroplasia; and 6) multiple hereditary osteochondromata.

A brief review of the clinical, laboratory, and pathological findings is presented with each disease entity.

Achondroplasia. Synonyms: chondrodystrophia fetalis, micromelia, chondrodystrophic dwarfism, chondrodystrophy.

Achondroplasia is perhaps the most familiar and certainly the most common of the cartilaginous dysplasias.

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